

BIOTERRORISM READINESS IN THE HEALTHCARE SETTING

Chelsea Romans, B.S.

INTRODUCTION

A bioterrorism attack is the deliberate release of viruses, bacteria, or other agents used to cause illness or death in people, animals, or plants. These agents are typically found in nature, but it is possible they could be modified to (1) increase their ability to cause disease, (2) make them resistant to current medicines, or (3) increase their ability to be spread into the environment. Terrorists may use biological agents because they can be extremely difficult to detect and do not cause illness for several hours to several days. Some bioterrorism agents, such as the smallpox virus, can be spread from person to person; other biological agents, such as anthrax, cannot be spread from person to person.

The Association for Professionals in Infection Control and Epidemiology (APIC) recognizes the importance of awareness of and preparation for bioterrorism on the part of healthcare facilities. Hospitals and clinics may have the first opportunity to recognize and initiate a response to a bioterrorism-related outbreak. Both federal and state public health agencies have programs in place to increase awareness regarding signs of bioterrorism among healthcare professionals.

Relying heavily on recent data from the Centers for Disease Control and Prevention (CDC), this article reviews the epidemiologic features of each of the category A (highest risk) agents. Infection control practices for patient management are outlined.

THREAT CLASSIFICATION

The CDC has a wealth of information regarding bioterrorism, which begins with a risk classification according to threat level (categories A, B, and C); category A threats are the biggest threats, and category C threats are the least threatening to people.¹ All biological agents fit into one

of these three categories. Category A agents are deemed to be “high priority” by the CDC because they can be easily spread among people; have unusually high fatality rates once infection occurs, and thus have the potential to cause panic in the public; and require specific, pre-planned public health action to manage large-scale infection. Category B agents are less of a threat, because they are defined as having lower rates of infection from person to person, have lower fatality rates, and require less emergency planning from organizations such as the CDC. Category C agents belong to the lowest threat category due to the difficulty of spread among people, but these agents are still of concern because of their wide availability to research laboratories.

Six biological agents are deemed to be high priority, or category A. This category includes: (1) anthrax (*Bacillus anthracis*), (2) botulism (*Clostridium botulinum* toxin), (3) plague (*Yersinia pestis*), (4) smallpox (*Variola major*), (5) tularemia (*Francisella tularensis*), and (6) viral hemorrhagic fevers (including filoviruses, arenaviruses, bunyaviruses, and flaviviruses). To understand the threat of category A agents, it is crucial to understand the process of infection, the symptoms and clinical diagnosis, possible modes of dissemination and disease spread, recommended treatments, and, finally, the mortality rate of each agent.

ANTHRAX

The bacteria *B. anthracis*, better known as anthrax, is a serious threat to public health when it is used as a biological weapon. To understand why this bacteria has the potential to be so dangerous, a basic understanding of the bacteria itself is necessary. Some bacteria, specifically the *Bacillus* and *Clostridium* genera, form structures called endospores when living in harsh temperature, chemical, or pressure environments. These complexes are dormant and very resistant to virtually any environmental condition. Endospores are nearly indestructible to conditions such as extreme heat, pressure, drying, chemicals, starvation, and antibiotics. Endospores can exist in this dormant state for thousands of years. Endospores become active when they are returned to favorable environmental conditions.

B. anthracis is ideal as a biological weapon because the spores are produced when the bacteria is in a harsh envi-

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ronment. These complexes can be sent via mail (e.g., the 2001 anthrax attacks on media news and senators' offices), where survival of the extremely resistant endospore structures makes this agent a very difficult weapon to combat. Once spores come in contact with humans, they can be inhaled or ingested, which gives the spores the favorable conditions needed to allow the bacteria to infect its host. Once in favorable conditions, the bacteria produce two main toxins—edema toxin and lethal toxin. These toxins enter target cells through the binding of protective antigen, which facilitates toxin entry. Edema toxin causes massive local edema. Lethal toxin causes the release of cytokines from the host macrophages, which is responsible for the sudden death seen in many anthrax patients.² Anthrax presents in three different forms: cutaneous anthrax, gastrointestinal anthrax, and pulmonary anthrax. Each type of anthrax infects its host through a different mechanism.

CUTANEOUS ANTHRAX

This form of anthrax is contracted when there is direct contact between *B. anthracis* or its endospores and a cut or abrasion in the skin. These openings then become red or raised and are often described as resembling an insect bite. However, within 2 to 14 days, the affected area becomes a painless open wound, which becomes black at the center. Two weeks from subsequent blackening of the wound, a scab is formed and falls off the skin. Symptoms include lymphangitis and painful lymphadenopathy, headache, and fever. The CDC recommends a variety of procedures to positively identify cutaneous anthrax; two separate swabs of the lesion (regardless of the stage) for a Gram's stain and culture, polymerase chain reaction (PCR) testing, biopsy of the infected lesion, ulcer, or scab, serum collection, and blood tests.³

Cutaneous anthrax is most often spread unintentionally, because many animals contain anthrax spores in their hooves, hides, hair, or wool; this form of anthrax is treated with antibiotics. Because the endospores are viable for a 60-day period, antibiotics are generally administered for 60 days. Approximately 95% of anthrax cases are of the cutaneous variety. Treated cutaneous anthrax cases very rarely result in death (less than 1%). Twenty percent of untreated cutaneous anthrax cases result in fatality.⁴

GASTROINTESTINAL ANTHRAX

Gastrointestinal anthrax (GI anthrax), is most often contracted through eating undercooked meat. Gastrointestinal exposure causes ulcers at the initial site of bacterial deposition, often the mouth or esophagus. Ulcers can also form in the intestines, mainly in the terminal ileum or cecum. Symptoms of GI anthrax include nausea, vomiting, bloody diarrhea, abdominal pain, and sepsis. GI anthrax, like cutaneous anthrax, is treated with a regimen of antibiotics. Untreated GI anthrax is estimated

to have a 25%–60% mortality rate.⁴ The effect of early antibiotic treatment is unknown, because there have been very few GI anthrax cases documented and studied.

PULMONARY ANTHRAX

Pulmonary anthrax, or inhalational anthrax, is by far the most fatal of the three different forms. To understand why inhalational anthrax is the highest threat of the three anthrax types, it is important to understand the molecular mechanism by which *B. anthracis* is infectious. After *B. anthracis* is inhaled, the alveolar macrophages begin to attack the spores, as would occur in any other normal immune response. However, rather than being eliminated by these macrophages, the spores survive within the alveolar macrophages and travel on regular routes of the macrophages—including the mediastinal lymph nodes and bloodstream. After the spores are deposited in the lymph nodes, *B. anthracis* can become active because conditions are favorable for the spores to thrive. More simply stated, the dormant form of the bacteria survives within the cells that are supposed to kill intruders. Further, not only do the endospores survive, but they travel within the macrophages and become active in organ systems that allow them to be circulated throughout the body.

Symptoms of inhalational anthrax can be broken into two separate and distinct stages. The first stage of infection is characterized by symptoms that are similar to those of a cold: mild fever, sore throat, coughing, vomiting, sweating, and muscle aches. After 2 days of these flulike symptoms, the second stage of infection occurs, which is marked by an intense fever, severe breathing problems, and shock. These symptoms occur as the disease progresses. Even with all possible medical support, inhalational anthrax fatality rates reach nearly 75%.⁴

BOTULISM

Botulism is caused by the gram-positive obligate anaerobic bacteria *C. botulinum*, which produces neurotoxins (toxins specifically affecting nerve cells) that enter the cytosol, and cleave proteins within the neuroexocytosis apparatus.⁵ Because acetylcholine—the neurotransmitter of both the peripheral nervous system and the central nervous system—cannot be released, muscle paralysis occurs. *C. botulinum* produces a variety of different toxins, called toxins A, B, C, D, and E. Humans are only affected by toxins A, B, and E. Toxins C and D cause botulism in non-human organisms.⁵ *C. botulinum* is similar to *B. anthracis* in that it also produces endospores. Botulism is most often associated with canned food, because this is the most common mode of transmission. However, although *C. botulinum* spores can survive in canned food, botulinum toxin cannot withstand temperatures higher than 80°C for longer than 10 minutes.⁵

According to the World Health Organization (WHO), there are five types of naturally occurring botulism:

(1) foodborne botulism, (2) infant botulism, (3) wound botulism, (4) adult infectious botulism, and (5) inadvertent botulism.⁵ Foodborne botulism results from the ingestion of food containing botulism neurotoxins. Similarly, infant botulism is caused by the ingestion of *C. botulinum*, which colonize and produce the deadly toxin within the intestinal tract of infants. Infants are at increased risk for *C. botulinum* colonization because they lack *Clostridium*-inhibiting bile acids found in the normal adult intestinal tract. Epidemiologic studies have identified honey as a natural reservoir of *C. botulinum*.⁶

Botulism caused by a wound is the least common form of the infection; this condition occurs when *C. botulinum* comes into direct contact with an open wound and later produces neurotoxins that spread throughout the bloodstream. The WHO recognizes a fourth category, adult infectious botulism, which includes all adults afflicted by botulism that cannot be traced to a specific food source or contact between a wound and the bacteria. The literature concerning this type of botulism suggests that these infections may result from intestinal colonization, which allows *in vivo* production of the neurotoxin.⁵ Studies have shown that patients with adult infectious botulism are linked with gastrointestinal surgeries.⁵ It is theorized that these surgeries may allow *C. botulinum* to become part of the normal gut flora, which eventually produces the botulism neurotoxin.

Inadvertent botulism describes accidental infection by botulinum toxin. Inadvertent botulism is a more recently evolved form of botulism that stems from the discovery that the botulism toxin, when carefully isolated and purified, can be used as an effective and powerful medication. Injecting overactive muscles with minute quantities of botulinum toxin type A results in decreased muscle activity by blocking the release of acetylcholine from the neuron. This renders the muscle unable to contract for several months. Hence, intramuscular injections of botulinum toxin are used to treat muscle spasms, dystonias, and other movement disorders. In cosmetic medicine, a small dose of botulinum toxin (i.e., Botox®, Allergan, Irvine, California) is used to prevent the formation of wrinkles by paralyzing facial muscles. Inadvertent botulism can occur due to accidental or overdose injections during the course of these treatments.

The *infective dose* of botulism neurotoxin is exceptionally small—only a few nanograms of toxin. (As a point of reference, a paper clip weighs approximately 1 g. There are 1,000,000,000 ng in 1 g.)

Botulism is commonly misdiagnosed, usually as a polyradiculoneuropathy (Guillain-Barré or Miller-Fisher syndrome), myasthenia gravis, or other central nervous system diseases.^{7,8} Symptoms of botulism tend to be neurologically linked, as the toxin affects acetylcholine production. Common symptoms are difficulty swallowing, speaking, or seeing, ptosis, diplopia (double vision), blurred vision, enlarged and/or delayed pupil reactions, dysarthria, dysphonia, dysphagia with a loss of

gag reflex, and paralysis. Paralysis begins at the cranial nerves and subsequently affects the upper extremities, respiratory muscles, and finally the lower extremities in a proximal-to-distal pattern.⁹ Communication becomes difficult—not because of disorientation—but rather due to prominent bulbar palsies, which cause weakness of the muscles innervated by cranial nerves of the lower brainstem. It is important to note that the patient does not have a fever; and because the toxin does not penetrate brain parenchyma, the patient does not become confused or disoriented. Clinically, the “classic triad” of botulism symptoms appears 1 to 2 days after infection and include symmetric, descending, flaccid paralysis with prominent bulbar palsies, afebrile state and clear sensorium.⁷ Also useful in the clinical diagnosis of botulism are the previously mentioned symptoms—diplopia, dysarthria, dysphonia, and dysphagia⁸—which are referred to as the “4 Ds” of the bulbar palsies.

Clinical diagnosis is different depending on the type of botulism infection, but all types require stool samples to either confirm or rule out botulism. In foodborne cases, laboratory confirmatory tests take days, so diagnosis should initially be made on the basis of patient history and physical findings. Patient history is significant because home canning and home pasteurization (particularly of vegetables) are leading causes of botulism in the United States.¹⁰ In suspected cases, serum, stool, and suspicious food should all be tested for the presence of botulism. Wound botulism is diagnosed through obtaining a sample or swab of the ulceration for anaerobic culture, as well as testing fecal samples (to rule out colonization in the intestine). Infant botulism is confirmed through stool samples; this condition should be suspected with diminished crying, sucking, and feeding as well as weakness in the neck and peripheral muscles. Stool samples also confirm adult infectious botulism and inadvertent botulism.

Botulism can be disseminated in a variety of ways, qualifying it as a possible agent of bioterrorism. Botulism can infect patients orally, inhalationally, parenterally, and dermally. Inhalational botulism has been proved possible by studies in which monkeys were infected with aerosolized botulism toxin.⁹ The toxin is absorbed in the lung tissue. Three cases of botulism in laboratory workers have been attributed to inhalation.¹¹ Botulism can be disseminated through dermal contact, as in wound botulism. Several cases of botulism due to therapeutic injections have been reported. Botulism is not communicable; that is, it is not transferable from person to person. Infection occurs only in persons who come directly in contact with the bacteria, spores, or neurotoxin.

The death rate from botulism between 1910 and 1919 was nearly 70%, but has since dropped to around 2% (in the 1990s); this reduction is mainly due to the advent of artificial respirators.¹² Although the mortality rate for treated botulism is approximately 2%, mortality rates for untreated cases of botulism are probably very high; how-

ever, because the treatment of botulism is standard and effective, concrete mortality rates for untreated botulism cases are not readily available. These fatality rates may seem unimpressive for one of the CDC's category A agents; however, the danger lies not in communicability, but in the machinery required for effective treatment. For example, according to a 2005 study, there were 2,688 full-feature ventilators available for use in New York City.¹³ According to the New York City Department of City Planning, the population of New York City was 7,956,113 in 2005.¹⁴ This means that if there were a bioterrorist attack on New York City that used aerosolized botulism as a weapon, many affected individuals would not receive proper medical care due to the unavailability of treatment (i.e., artificial respirators).

PLAGUE

Plague, caused by the bacteria *Y. pestis*, has a prominent place in world history; this biological agent has been attributed with causing three pandemics in the last 1500 years.¹⁵ Given the enormous potential for damage that this bacteria carries, combined with the high level of communicability of the disease, the CDC classifies *Y. pestis* as a category A threat. In nature, plague is caused by the bite of infected fleas. Naturally occurring outbreaks of plague are rare due to improved sanitation standards, which reduce the amount of rodents and thereby reduces the amount of infected fleas. In humans, *Y. pestis* causes three different forms of infection: pneumonic, septicemic, and bubonic. Generally, the mechanism by which plague kills people is the migration of bacteria through the lymph nodes. The bacteria are phagocytosed (engulfed by cells of the immune system), but they are not destroyed. The bacteria then quickly multiplies and causes destruction and necrosis of the lymph node architecture, which eventually causes bacteremia, septicemia, and endotoxemia. Each form of the infection has a different process of infection, varied symptoms, and separate modes of dissemination.

PNEUMONIC PLAGUE

Pneumonic plague is the most fatal form of the infection, but it accounts for fewer than 14% of all naturally occurring plague cases.¹⁶ Pneumonic plague is acquired by inhaling droplets containing the bacteria, which infect the lungs. Approximately 5% of pneumonic plague is attributed to cases of bubonic plague, which if left untreated, will infect the lungs of the patient.¹⁷ There is a 2- to 3-day incubation period, after which the patient presents with initial flulike symptoms that include fever, headache, and weakness. Progression to chest pain, difficulty breathing, and hemoptysis follow. Pneumonia rapidly develops, and within 2 to 4 days of pneumonia onset, respiratory failure and shock may occur. Nausea, vomiting, and abdominal pain may also occur.

Pneumonic plague should be suspected if a smear taken from tracheal or lung aspirate indicates the presence of gram-negative coccobacilli bacteria. The CDC confirms pneumonic plague with the presence of one of the following indicators¹⁸:

- * If a culture is lysed by specific bacteriophage.
- * If two serum specimens demonstrate a fourfold anti-F1 antigen titer difference by agglutination testing.
- * If a single serum specimen tested by agglutination has a titer of > 1:128, and the patient has no known previous plague exposure or vaccination history.

Chest x-ray showing fulminating pneumonia and culture of sputum are also used to confirm a *Y. Pestis* infection.¹⁷ Diagnosis is typically based on clinical presentation and is confirmed through laboratory testing within 24 to 48 hours.

BUBONIC PLAGUE

Bubonic plague—the version of the *Y. Pestis* infection that is well known for the mass destruction it caused in Europe during the Middle Ages—is caused by the bite of an infected flea. As previously mentioned, the risk of another large-scale bubonic plague infection is limited by improved sanitation practices in many large cities. Bubonic plague symptoms include fever, headache, and malaise, which are followed by the development of swollen and painful lymph nodes called *buboes*. Symptoms typically occur 2 to 6 days after exposure. When bubonic plague is left untreated, the bacteria eventually infect the bloodstream (i.e., septicemic plague), multiply, and cause shock. Diagnosis of bubonic plague follows the diagnosis protocol of pneumonic and septicemic plague, as specified by the CDC.

SEPTICEMIC PLAGUE

Septicemic plague describes the condition in which the bacteria *Y. Pestis* multiplies in the blood and causes toxemia. This form of plague is capable of causing disseminated intravascular coagulation, which is similar to other types of gram-negative sepsis. Septicemic plague can be a result of the other types of plague infection (pneumonic, bubonic), or it can occur by itself. Septicemic plague is most often a result of bubonic plague. When it occurs as a primary infection, septicemic plague is contracted through the bite of an infected organism, or by infected material breaking through the patient's skin. Although the mechanism of contracting septicemic plague is the same as that of bubonic plague, patients do not develop buboes. Similar to bubonic plague, septicemic plague is not communicable.

Symptoms include acute onset of fever, chills, prostration, abdominal pain, nausea, vomiting, bleeding into the skin, shock, seizures, confusion, red or black rash on the

skin, and blackening of the extremities.¹⁹ Diagnosis of septicemic plague can be difficult, because it has symptoms similar to lymphogranuloma venereum, shigellosis, syphilis, hernia, cat scratch fever, typhoid fever, and appendicitis. Effective diagnosis of septicemic plague involves patient history, auscultation, and ruling out of diseases with similar symptoms. *Y. Pestis* infections are confirmed in laboratories, because the bacteria is known to be a non-motile, gram-negative bacillus that shows bipolar staining with select stains. There are a variety of other microbiological laboratory techniques used to confirm the presence of the bacteria; however, treatment should proceed if suspicion is strong that a patient does have plague, because it progresses quickly.²⁰

PLAGUE TREATMENT

Treatment for plague is generally understood to be effective only within 24 hours of the first symptoms. After the first 24 hours pass, the efficacy of any treatment option is greatly reduced.¹⁹ All forms of plague are treated with antibiotics, including streptomycin, gentamicin, doxycycline, and ciprofloxacin. Because pneumonic plague can be transferred from person to person via droplets, these patients are kept in strict isolation during the antibiotic treatment. Mortality rates for untreated pneumonic plague are nearly 100%; antibiotic treatment within 24 hours of symptom presentation reduces mortality rates to approximately 50%. Untreated bubonic and septicemic plague have mortality rates between 50% and 90%; treatment reduces mortality rates to approximately 1%-15%.²¹

Plague is a likely candidate in a biological attack for a variety of reasons, including the widespread availability of *Y. Pestis* in microbe banks worldwide (as well as its rather common natural availability); the relative ease of aerosolization; the short length of time between dissemination and death; the ability of pneumonic plague to be spread from person to person after the initial infection; and the very high mortality rate, particularly in pneumonic and septicemic cases.²²

SMALLPOX

The term *smallpox* was first used in Europe during the 15th century to distinguish this disease from the "great pox" (syphilis).²³ Smallpox is an acute contagious disease caused by one of two variants of the *Variola* virus: *Variola major* (responsible for approximately 90% of smallpox cases) or *Variola minor*.²⁴ *Variola major* is the more severe and most common form of smallpox that presents with a more extensive rash and high fever.

There are four types of *Variola major* smallpox: *ordinary*, *modified*, *hemorrhagic*, and *malignant* (or flat). Ordinary and modified are the two most common types and are associated with a mortality rate of approximately 30%.²⁴ The hemorrhagic and malignant varieties are

rare and more lethal. Hemorrhagic smallpox is invariably fatal, characterized by hemorrhage in the mucous membranes and skin (along with the typical skin rash). Malignant smallpox is characterized by a flat rash. Malignant smallpox also has an incredibly high mortality rate. Smallpox caused by one of the *Variola major* viruses is frequently fatal; once a person has contracted the disease, no effective treatment exists. The only healthcare strategy is prevention through vaccination. Smallpox was officially eradicated (as endorsed by the World Health Assembly) in 1980 after millions of people worldwide received the smallpox vaccination.

Variola minor is a far less common presentation of smallpox, and a much less severe disease, with historical death rates of 1% or less.²⁴

Smallpox symptoms typically occur 12 to 14 days after a patient comes in contact with the virus. During this incubation period, patients appear and feel healthy, and they are not infectious. The incubation period is followed by flulike symptoms that include fever, malaise, headache, and prostration. Occasionally, patients also experience vomiting and abdominal pain. Three days after the onset of these initial symptoms, body temperature drops so that the patient is relieved of the flulike symptoms. However, the patient then develops a rash on the face, hands, and forearms. The rash eventually progresses to the trunk. Lesions also develop in the nose and mouth and quickly intensify, releasing large amounts of virus into the mouth and throat. Lesions progress from macules to papules to vesicles to pustules. All lesions in a given area progress together through these stages. From 8 to 14 days after the onset of symptoms, the pustules form scabs that leave depressed depigmented scars upon healing.²⁵ According to the CDC, the clinical case definition of smallpox is "an illness with acute onset of fever $\geq 101^{\circ}\text{F}$ (38.3°C), followed by a rash characterized by firm, deep-seated vesicles or pustules in the same stage of development without other apparent cause."²⁶

Smallpox can be confirmed in the laboratory through PCR. Laboratory confirmation is only performed at CDC laboratories. Because smallpox has been eradicated, a single naturally occurring case would be considered an outbreak. The CDC has listed the criteria for suspected, probable, and confirmed cases as follows:

Confirmed case: A case of smallpox that is laboratory confirmed, or a case that meets the clinical case definition that is epidemiologically linked to a laboratory confirmed case.

Probable case: A case that meets the clinical case definition, or a case that does not meet the clinical case definition but is clinically consistent with smallpox and has an epidemiologic link to a confirmed case of smallpox. Examples of clinical presentations of smallpox that would not meet the ordinary type of clinical case definition are: (1) hemorrhagic type, (2) flat type, and (3) *variola sine eruptione*.

Suspect case: A case with a febrile rash illness, with fever preceding development of rash by 1 to 4 days.²⁶

Smallpox is an intensely contagious disease. The rates of transmission seem to be highest in face-to-face interaction during the first week of the rash, as the virus is being released through the respiratory tract. Patients remain infectious until the last scab from the pustule has fallen off; however, the virus contained in the pustules is not highly infectious. It seems that without any immunity in the form of vaccination, humans are invariably susceptible to the *Variola* virus. Humans are the only reservoir of smallpox; there is no animal or insect vector. During the eradication campaign, investigation showed that the virus seemed to be able to spread through building ventilation systems. Some experts have estimated today's rate of transmission to be on the order of 10 new infections per infected person.²⁵

The only treatment shown to be effective for smallpox is the vaccine, which contains live virus from the *Orthopoxvirus* genus (a closely-related viral genus). Vaccination can be administered up to 4 days after exposure to reduce symptoms and mortality rates. The smallpox vaccine provides protective immunity and can lessen the severity of the disease. Currently, the antiviral medication cidofovir is being tested in laboratories as a treatment for smallpox, and the initial results have been promising. As mentioned earlier, mortality rates are typically around 30% for the more common smallpox infections, with the hemorrhagic and malignant smallpox infections having a much higher mortality rate of nearly 100%.²⁵

Smallpox has been flagged as a danger for use as a biological weapon because of its ability to be transmitted from person to person with relative ease, its ability to be aerosolized, and also because of the limited availability of the vaccine.

TULAREMIA

Tularemia is an infectious, non-contagious bacterial disease caused by the bacterium *F. tularensis*. This disease mainly affects animals—particularly rabbits, rodents, and hares. It has been reported across the United States, except for Hawaii. Tularemia is considered to be one of the most pathogenic agents known and is thus categorized as category A. Persons most often become infected with *F. tularensis* through the bite of an infected insect, especially ticks and deerflies; however, infection can also occur through handling infected animals, eating infected food, or breathing bacteria that are airborne. *F. tularensis* has been classified into four different subspecies: *tularensis*, *holarctica*, *mediasiatica*, and *novicida*. *F. tularensis*, also called type A, has the highest mortality rates of the subspecies.

There are several types of tularemia, including ulceroglandular tularemia, glandular tularemia, oculoglandular tularemia, oropharyngeal tularemia, pneumonic tularemia, and typhoidal tularemia. The incubation period

for *F. tularensis* is between 2 and 10 days. Ulceroglandular tularemia, which occurs after a patient has been bitten by an infected animal or insect, presents with symptoms that include a skin ulcer at the site of the infected insect or animal bite, swollen and painful lymph nodes, fever, chills, headache, and exhaustion. Glandular tularemia has a nearly identical symptom list, except for skin ulcers. Oculoglandular tularemia is typically acquired when a person with infected hands rubs the eyes, thereby passing the bacteria into the eyes; this infection causes eye pain, eye redness and swelling, and an ulcer on the inside of the eyelid. Oropharyngeal tularemia—caused by the consumption of undercooked, infected meat—affects the digestive tract and causes vomiting and diarrhea. Pneumonic tularemia, caused by the inhalation of *F. tularensis*, causes symptoms typical of pneumonia, including cough, chest pain, and difficulty breathing. The inhalation of only 10 to 50 organisms can cause tularemia in humans.²⁷ Typhoidal tularemia is marked by fever, exhaustion, and weight loss. This type of tularemia may affect a number of organs in the body, most notably of which are the lungs.

Humans become infected through the bites of infected animals and insects. It is also common for humans to be infected through contaminated meat and water supplies. Tularemia is a category A agent because of the extremely low infective dose; its ability to survive in water, soil, carcasses, and frozen meats; and the relative ease with which it can be aerosolized. The United States has included tularemia in its biological warfare program and has conducted tests of the weaponized bacterium during the 1950s and 1960s. Other countries are also thought to have *F. tularensis* currently available as a biological weapon.

Tularemia can be treated with antibiotics. Typically, streptomycin and tetracycline are prescribed, with gentamycin being a less common but still effective treatment option. Five percent of untreated tularemia cases are fatal; only approximately 1% of treated cases are fatal.²⁷

VIRAL HEMORRHAGIC FEVERS

Viral hemorrhagic fevers (VHFs) describe a group of viruses that affect multiple organ systems in the body.²⁸ VHFs include four different families of viruses: (1) arenaviruses, (2) filoviruses, (3) bunyaviruses, and (4) flavaviruses.²⁹ Some diseases caused by these families of viruses are categorized together due to several distinct characteristics (Table 1). VHF viruses are generally zoonotic (i.e., affecting animals who then transmit the viruses to humans). These viruses typically use animals (generally rodents and arthropods) as reservoirs and insects as vectors. VHFs are transmitted to humans when infected virus reservoirs come into contact with unaffected humans. This contact usually occurs in the form of handling the urine, feces, saliva, or other body excretions from infected animals. Transmission also occurs

TABLE 1. Symptoms, Modes of Transmission and Mortality Rates for VHF's

	Disease (Virus)	Usual Vector	Incubation Period (Days)	Symptoms
Arenaviridae	Lasza Fever ^{30,33}	Rodent	5-21	80% of cases are asymptomatic; the remaining cases present with the following symptoms: fever; retrosternal pain, sore throat, back pain, cough, abdominal pain, vomiting, diarrhea, conjunctivitis, facial swelling, proteinuria, and mucosal bleeding Neurological problems have also been documented: hearing loss, tinnitus and encephalitis
	Argentine HF (Junin) ^{30,33}	Rodent	7-16	Fever, eye redness, fatigue, dizziness, muscle aches, loss of strength, exhaustion; bleeding under skin, internal organs, or from body orifices, and shock
	Bolivian HF (Machupo) ^{30,33}	Rodent	7-16	Fever, malaise, headache, muscle pains, hemorrhages on margins of gums Approx. 1/3 of patients develop delirium, convulsions, and serious hemorrhages (may include petechiae, or bleeding from nose, gums, or intestine).
Arenavirus	Brazilian HF (Sabia) ^{30,33}	Rodent	7-16	Fever, eye redness, fatigue, dizziness, muscle aches, loss of strength, exhaustion, hemorrhaging from body orifices, shock, nervous system malfunction, coma, delirium and seizures
	Venezuelan HF (Guanarito) ^{30,33,34}	Rodent	7-16	Fever, malaise, headache, bleeding gums, arthralgia, sore throat, vomiting, abdominal pain and myalgia
Bunyaviridae				
Phlebovirus	Rift Valley Fever ^{30,35}	Mosquito	2-6	Mild form: fever, muscle pain, joint pain, headache Severe form – Ocular form: "Mild form" symptoms, in addition to retinal lesions Severe form – Meningoencephalitis form: "Mild form" symptoms in addition to severe headache, loss of memory, hallucinations, confusion, vertigo, disorientation, lethargy, coma Severe form – Hemorrhagic form: "Mild form" symptoms in addition to severe liver impairment (e.g., jaundice), vomiting blood, passing blood in feces, purpura, bleeding from nose or gums, menorrhagia
Nairovirus	Crimean-Congo HF ^{30,36}	Tick	3-12	High fever, headache, back pain, joint pain, stomach pain, vomiting, red eyes, flushed face, Petechiae on the palate, jaundice, changes in mood and sensory perception, large areas of severe bruising, severe nose bleeds
Hantavirus	Hantavirus pulmonary syndrome (HPS) HF with renal syndrome (HFRS) ^{30,37}	Rodent	9-35	HPS symptoms: fever, headache, stomach pain, back pain, joint pain, difficulty breathing, coughing, nausea, vomiting HFRS symptoms: intense headaches, back and abdominal pain; fever, chills, nausea, blurred vision
Filoviridae				
Filovirus	Marburg and Ebola ^{30,38-41}	Unknown	Ebola: 2-21 Marburg: 2-14	Marburg: fever, chills, headache, myalgia, maculopapular rash, nausea, vomiting, chest pain, sore throat, abdominal pain, diarrhea, jaundice, inflammation of parotids; severe weight loss, delirium, shock, liver failure, multi-organ dysfunction Ebola: fever, headache, joint and muscle aches, sore throat, diarrhea, vomiting, stomach pain, rash, red eyes, hiccups, internal and external bleeding
Flaviviridae				
Flavivirus	Yellow Fever ^{30,42}	Mosquito	3-6	Fever, chills, severe headache, back pain, general muscle aches, nausea, fatigue, weakness, hematemesis, epistaxis, gum bleeding, purpuric hemorrhages, jaundice, hypotension, shock, metabolic acidosis, acute tubular necrosis, myocardial dysfunction, cardiac arrhythmia
	Dengue HF ^{30,43}	Mosquito	Unknown	Decreased appetite, fever, headache, joint aches, malaise, muscle aches, vomiting, ecchymosis, generalized rash, Petechiae, shock-like state, red eyes, enlarged liver, low blood pressure

TABLE 1. (Continued)

Treatment	Mortality Rates-Treated	Mortality Rates-Untreated	Contagious?
Ribavirin Vaccination is available Treatment includes convalescent human plasma	1-2% 1-2%	15-25% 15-30%	Yes—through aerosolized particles, contact with bodily fluids, possibly including sexual contact Yes—through contact with bodily fluids
Ribavirin		30%	Debated, but CDC lists Machupo as transmissible from person-to-person
Ribavirin		Only 3 reported cases Estimated mortality rate is 33%	Yes—through bodily fluids
Ribavirin, mechanical ventilation, dialysis Ribavirin is also used		25%	Unknown
The mild form of RVF requires no treatment as it generally runs its course quickly and without lasting effects. The treatment for the severe forms entails general supportive therapy.		Approximately 1% in the mild, ocular, and meningoencephalitis forms Approximately 50% in the hemorrhagic (also the rarest) form	None have been demonstrated, but there is a theoretical risk to healthcare workers through infected blood and tissues
Primarily supportive care Ribavirin is also used	No data	Ranges from 9-50% in documented outbreaks	
HPS treatment: Mechanical ventilation, intravenous Ribavirin has shown to be somewhat effective HFRS treatment: Typically involves supportive therapy; intravenous Ribavirin is also used.	HPS: Estimated 39% HFRS: 1-15%	HPS: Possible, but not confirmed HFRS: May occur, but is extremely rare	
No standard treatment for Marburg or Ebola Supportive therapy is used.		Marburg: Estimated 23-93% Ebola: 50-89%	
Vaccination is available	Varies from 15-50%	Varies from 15-50%	Yes
Supportive treatment only	Most patients recover. However, half of all patients who go into shock do not.		Yes

in the form of bites by infected arthropods to humans. VHF's can also be transmitted from an arthropod (tick or mosquito) bite to an animal, and then from the infected animal to a human. Some of these viruses can be transmitted among people, whereas some can also be transmitted through close contact with body fluids. Table 1 expresses the symptoms, modes of transmission, and mortality rate for each of the VHF's.

REPORTING REQUIREMENTS

Healthcare facilities may be the initial site of recognition and response to bioterrorism events. Should clinicians suspect a bioterrorism event, the healthcare facility administration should be notified immediately so that emergency response systems can be activated. The healthcare facility administration will initiate a network of communication that involves local infection control personnel, local and state health departments, the Federal Bureau of Investigation (FBI) field office, the CDC, and medical emergency services.

INFECTION CONTROL PRACTICES FOR PATIENT MANAGEMENT

The epidemiologic features of the specific bioterrorism agent will dictate the specific patient management practices. The infection control practices for standard precautions, droplet precautions, airborne precautions, and contact precautions should be familiar to all healthcare workers. Only a brief text and table review of these practices are provided here (Table 2).

STANDARD PRECAUTIONS

As stated earlier, many agents of bioterrorism are not transmitted from person to person. Therefore, many patients with suspected or confirmed bioterrorism-related illnesses can be managed using standard precautions. Standard precautions are used for all patients receiving care, regardless of their diagnosis or presumed infection status; these precautions are designed to reduce transmission from both recognized and unrecognized sources of infection. Standard precautions prevent direct contact with all body fluids, secretions, excretions, non-intact skin (including rashes), and mucous membranes. Standard precautions include handwashing and the use of gloves, masks, eye protection, and/or gowns when performing patient care activities that may result in splashes of body fluids.

DROPLET PRECAUTIONS

Droplet precautions are used for patients known or suspected to be infected with microorganisms transmitted by large particle droplets, generally larger than 5 microns

in size. These droplets can be generated by the infected patient during coughing, sneezing, talking, or during respiratory-care precautions. Droplet precautions require healthcare providers to wear a surgical-type mask when they are within 3 feet of the infected patient.

AIRBORNE PRECAUTIONS

Airborne precautions are used for patients known or suspected to be infected with microorganisms that are smaller than 5 microns and can remain suspended in air. These microorganisms can be widely dispersed by air currents. Airborne precautions require healthcare providers to wear respiratory tract protection when entering the patient's room. Patients confined to airborne precautions must be housed in a room that meets specific ventilation and engineering requirements; the door must remain closed.

CONTACT PRECAUTIONS

Contact precautions are used for patients known or suspected to be infected with epidemiologically important organisms that can be transmitted by direct contact with the patient or indirect contact with potentially contaminated surfaces in the patient's care area. Contact precautions require healthcare providers to: (1) wear clean gloves upon entry into the patient's room; (2) wear a gown for all patient contact and for all contact with the patient's environment (the gown must be removed before leaving the patient's room); and (3) wash hands using an antimicrobial agent.

A component of contact precautions is the careful management of potentially contaminated equipment and environmental surfaces. When possible, noncritical patient care equipment should be dedicated to a single patient (or cohort of patients with the same illness). If the use of common items is unavoidable (such as x-ray equipment), all potentially contaminated equipment should not be used for the care of another patient until it has been appropriately cleaned and reprocessed. All surfaces should be cleaned using either an Environmental Protection Agency-registered, facility-approved sporicidal/germicidal agent or 0.5% hypochlorite solution (one part household bleach added to nine parts water).

CONCLUSION

Healthcare facilities may be the initial sites of recognition and response to bioterrorism event. Therefore, the awareness and preparation for bioterrorism on the part of hospitals and clinics is essential. Healthcare facilities should have infection control policies in place to rapidly implement prevention and control measures in response to a suspected outbreak. The first step in this effort is to understand basic facts regarding the six biological agents that are deemed to be high priority, or category A, by the CDC.

TABLE 2. Agent-Specific Recommendations for Patient Management⁴⁴⁻⁴⁶

Anthrax⁴⁴	
Isolation	Not necessary; follow standard precautions.
Patient placement	A private room is not necessary.
Patient transport	Follow standard precautions.
Cleaning of equipment/environment	Follow standard cleaning procedures.
Notes	Person-to-person transmission is very unlikely. Airborne transmission does not occur, but direct contact with skin lesions may result in skin infection.
Botulism⁴⁴	
Isolation	Not necessary; follow standard precautions.
Patient placement	A private room is not necessary.
Patient transport	Follow standard precautions.
Cleaning of equipment/environment	Follow standard cleaning procedures.
Notes	Person-to-person transmission does not occur. Even a single case should raise concerns of an outbreak associated with shared contaminated food.
Plague: pneumonic⁴⁴	
Isolation	Yes; follow standard and droplet precautions.
Patient placement	Private room or place with cohort of other patients with similar symptoms and same presumptive diagnosis. Special air handling is not necessary, and doors may remain open.
Patient transport	Limit movement; when transport is unavoidable, follow droplet precautions. Place a surgical mask on patient to minimize dispersal of droplets.
Cleaning of equipment/environment	Clean equipment and surfaces according to contact precautions procedures. Handle contaminated clothing minimally to avoid agitation. Store clothing in labeled, plastic bags.
Notes	<i>Yersinia pestis</i> is usually transmitted by infected fleas, which results in lymphatic and blood infections (bubonic and septicemia plague). A bioterrorism-related outbreak may be expected to be airborne, causing a pulmonary variant (pneumonic plague).
Smallpox⁴⁴	
Isolation	Yes; follow standard, airborne, and contact precautions.
Patient placement	Place in private room that meets the ventilation and engineering requirements for airborne precautions.
Patient transport	Limit movement; when transport is unavoidable, follow contact and droplet precautions. Place a surgical mask on the patient to minimize dispersal of droplets.
Cleaning of equipment/environment	Clean equipment and surfaces according to contact precautions procedures.
Notes	Healthcare facilities without patient rooms appropriate for airborne precautions should have a plan for transfer of suspected or confirmed smallpox patients to a nearby facility with appropriate isolation rooms.
Tularemia¹⁰	
Isolation	Not necessary; follow standard precautions.
Patient placement	A private room is not necessary.
Patient transport	Follow standard precautions.
Cleaning of equipment/environment	Follow standard cleaning procedures.
Viral Hemorrhagic Fevers⁴⁶	
Isolation	Yes; follow standard, contact, and droplet precautions. Patients with respiratory tract symptoms should also wear a mask.
Patient placement	Place the patient in a private room.
Patient transport	Limit movement; when transport is unavoidable, follow contact and droplet precautions. Place a surgical mask on the patient to minimize dispersal of droplets.
Cleaning of equipment/environment	Clean equipment and surfaces according to contact precautions procedures.
Notes	Although transmission by an airborne route has not been established, hospitals may choose to use airborne precautions for patients who have severe pulmonary involvement.

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BIOTERRORISM READINESS IN THE HEALTHCARE SETTING POST TEST

Expires: October 15, 2012 Approved for 2 ARRT Category A Credits

1. **The CDC categories for classifying biological threats are**
 - a. one, two, three.
 - b. red, yellow, green.
 - c. A, B, C.
 - d. global, federal, local.
2. **Which of the following is NOT a characteristic of high-priority biological threats?**
 - a. They can be easily spread among people.
 - b. They have unusually high fatality rates once infection occurs.
 - c. They have the potential to cause panic.
 - d. They can result in rare diseases.
3. **How many biological agents has the CDC deemed to be "high priority"?**
 - a. 2
 - b. 6
 - c. 9
 - d. 12
4. **What bacteria cause the disease known as anthrax?**
 - a. *Bacillus anthracis*
 - b. *Bacillus cereus*
 - c. *Clostridium perfringens*
 - d. *Azotobacter vinelandii*
5. **Why is it significant that the bacterium that causes anthrax form endospores?**
 - a. Endospores are very tough, resistant structures that become active when they find favorable conditions, such as the human body.
 - b. Endospores are easily killed by fluctuation in temperature or exposure to moisture.
 - c. Endospores cannot be inhaled or ingested by humans, which makes transmission difficult.
 - d. Endospores are rapidly reproducing structures, although they live only briefly.
6. **Cutaneous anthrax is contracted when**
 - a. humans or animals eat undercooked meat contaminated by *B. anthracis*.
 - b. there is direct contact between *B. anthracis* or its endospore and a cut or abrasion in the skin.
 - c. *B. anthracis* is inhaled.
 - d. the skin of a healthy person comes in contact with *C. perfringens*, which is found on dead bodies.
7. **What is the most common form of anthrax?**
 - a. Gastrointestinal anthrax
 - b. Pulmonary anthrax
 - c. Cutaneous anthrax
 - d. Hemorrhagic anthrax
8. **Which form of anthrax is associated with the highest mortality rate?**
 - a. Gastrointestinal anthrax
 - b. Pulmonary anthrax

- c. Cutaneous anthrax
d. Hemorrhagic anthrax
9. **What cell type(s) is (are) targeted by the bacteria *Clostridium botulinum*?**
a. Nerve cells
b. Blood and immune system cells
c. Smooth muscle cells
d. Hormone-secreting cells
10. **What is the most common mode of transmission for botulism?**
a. Animals that contain the spores in their hooves, hides, or hair
b. Bites of infected fleas
c. Bites of infected mosquitoes
d. Canned food
11. **Which of the following is NOT a type of naturally occurring botulism?**
a. Foodborne botulism
b. Infant botulism
c. Airborne botulism
d. Inadvertent botulism
12. **Which is the least common form of *C. botulinum* infection?**
a. Foodborne botulism
b. Infant botulism
c. Wound botulism
d. Adult infectious botulism
13. **The botulism toxin, when carefully isolated and purified, can be used**
a. as a preservative in canned foods.
b. to treat muscle spasms, dystonias, and other movement disorders.
c. to discourage scar formation through direct application to the skin.
d. to treat gastrointestinal ulcers.
14. **Which of the following are common symptoms of botulism?**
a. Difficulty swallowing or speaking; blurred vision, paralysis
b. Difficulty breathing; arrhythmia, hypotension
c. Hematuria, painful urination
d. Raised rash on extremities confusion; disorientation
15. **The diagnosis of botulism is confirmed by**
a. blood cultures.
b. stool samples.
c. chest radiograph.
d. biopsy of the stomach lining.
16. **Which of the following is a TRUE statement regarding the use of botulism as a possible agent of bioterrorism?**
a. The *C. botulinum* bacteria cannot be aerosolized.
b. Botulism can only be transmitted to humans through contaminated food.
c. Botulism is easily transmitted from person-to-person.
d. Botulism can infect patients orally, inhaled, parenterally, and dermally.
17. **In the event of a bioterrorism attack on a major US city using *C. botulinum* bacteria, which resource would most likely be in short supply?**
a. Surgical masks
b. Antibiotics, such as fluoroquinolones
c. Artificial respirators
d. Isolation rooms that meet ventilation and engineering requirements for airborne precautions
18. **In nature, plague is caused by**
a. a retrovirus, transmitted to humans by infected cats.
b. the bacteria *Yersinia pestis*, spread by the bite of infected fleas.
c. the Machupo virus, spread by infected rodents.
d. a mosquito-borne Flavivirus.
19. **Generally, the mechanism by which plague kills people is**
a. migration of bacteria through the lymph nodes.
b. bacterial triggering of an intense immune system reaction.
c. localized bacterial-induced infection that results in a rapidly widening skin wound.
d. the collection of bacteria in the brain and spinal cord.
20. **Which is the most fatal form of plague?**
a. Bubonic
b. Septicemic
c. Pneumonic
d. No forms of plague are ever fatal.
21. **Buboes that accompany cases of bubonic plague are actually**
a. internal ulcerations.
b. rashes that are red and raised.
c. small purple or red spots on the skin that are caused by minor hemorrhage.
d. swollen and painful lymph nodes.
22. **In septicemic plague, the *Y. pestis* bacteria multiplies in the**
a. bone marrow.
b. lungs.
c. liver.
d. blood.
23. **Which of the following is a TRUE statement regarding septicemic plague?**
a. It can be a result of other types of plague infection, or it can occur by itself.
b. It is most often the result of consuming tainted food.
c. Patients develop multiple, painful buboes.
d. Symptoms are unique and characteristic, making diagnosis straightforward.
24. **To be effective, treatment for plague must begin within ___ of the symptom onset.**
a. 12 hours
b. 24 hours
c. 48 hours
d. 5 days

25. All forms of plague are treated with
- steroids.
 - antiviral medications.
 - antibiotics.
 - immunoglobulin.
26. The term smallpox was first used in the 15th century to distinguish this disease from the “great pox,” which is actually
- herpes zoster (shingles).
 - syphilis.
 - varicella (chickenpox).
 - diphtheria.
27. Smallpox symptoms typically occur _____ days after a patient comes in contact with the virus.
- 2 - 4
 - 6 - 8
 - 12 - 14
 - 16 - 20
28. Tularemia mainly affects
- infants.
 - pregnant women.
 - animals.
 - the elderly.
29. All of the following belong to the group of viruses known as viral hemorrhagic fevers EXCEPT
- arenaviruses.
 - filoviruses.
 - bunyaviruses.
 - rhinoviruses.
30. Which of the following is NOT required to comply with contact precautions?
- Wear clean gloves upon entry into the patient’s room.
 - Wear respiratory protection when entering the patient’s room.
 - Wear a gown for all patient contact and for all contact with the patient’s environment.
 - Wash hands using an antimicrobial agent.



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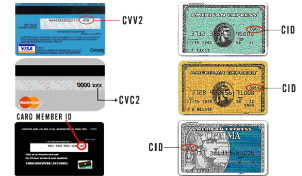
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2. a b c d	7. a b c d	12. a b c d	17. a b c d	22. a b c d	27. a b c d
3. a b c d	8. a b c d	13. a b c d	18. a b c d	23. a b c d	28. a b c d
4. a b c d	9. a b c d	14. a b c d	19. a b c d	24. a b c d	29. a b c d
5. a b c d	10. a b c d	15. a b c d	20. a b c d	25. a b c d	30. a b c d